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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/998,058	11/30/2001	David W. Threadgill	421/34/2	6701

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EXAMINER	
SAKELARIS, SALLY A	
ART UNIT	**PAPER NUMBER**
1634	

DATE MAILED: 09/07/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/998,058	THREADGILL ET AL.	
	Examiner	Art Unit	
	Sally A Sakelaris	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *06 July 2004*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-27,46-53 and 61-72* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-27,46-53 and 61-72* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submissions filed on 7/6/2004 have been entered.

This action is written in response to applicant's correspondence submitted 7/6/2004 Claims 1, 2, 11-18, and 46-47 have been amended, claims 28-45 and 54-59 have been canceled, and claims 60-72 have been added. Claims 1-27, 46-53, and 61-72 are pending. Applicant's amendments and arguments have been thoroughly reviewed, but are not persuasive for the reasons that follow. Any rejections not reiterated in this action have been withdrawn as necessitated by applicant's amendments to the claims.

Claim Interpretation

It should be noted that some of the art cited in this office action is intending to relay the breadth of the claims as currently written.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

1. Claims 1-10, 15, 19-27, and 46-53 are rejected under 35 U.S.C. 102(b) as being anticipated by Diehl et al, PNAS 1997.

Diehl et al. teach a method for identifying multiple genetic loci for example, *Col2a1, Col1a1* and *Col3a1*(page 5235) that modulate the phenotype of facial clefting in mice. Diehl et al have performed a genome-wide search for loci contributing to susceptibility to teratogen-induced facial clefting in the mouse. AXB and BXA recombinant inbred(RI) lines derived from crosses between A/J and C57BL6/J strains were supplied by M. Nesbitt and the mice were then bred by intercrossing recombinant inbred lines and maintained in a colony at the University of Michigan(page 5232) as a renewable population of genetically diverse individuals. The reference teaches this study for identifying a genetic locus in the diploid mouse system wherein the inbred lines of the renewable population of genetically diverse individuals comprise less than about 100 strains, in one instance a BXD set of 26 RI lines is used(page 5234). Experiments were also performed using the AXB and BXA RI strains to evaluate both spontaneous and teratogen-induced clefting resulting in both visual and physiological phenotypes. The reference uses the extensive data on teratogen-induced clefting in the AXB and BXA RI lines collected previously with a genome wide collection of marker typings for these RI lines to study the effects of genetic polymorphisms segregating in the renewable population(page 5232, left column). Diehl et al. teach the resulting molecular phenotype of their mouse mutants with clefting phenotypes to include for example, eight collagen genes including an altered expression of one, *Col3a1*, which is normally expressed in the embryonic palate. The reference also teaches the method for identifying multiple genetic loci further comprising identifying two or more

genetic loci that modulate the phenotype of clefting as seen on the reference's page 5235 in their explanation that in addition to *Col3a1*, two other genetic factors, *Col1a1* and a cyclic nucleotide phosphodiesterase gene are located on the same chromosome and are thought to together, be possibly relevant to the role of cAMP in the etiology of cleft palate abnormalities(page 5235). Additionally, the reference teaches the implication of the tenascin C gene, an extracellular matrix protein, and several cell-signaling molecules which have been previously implicated in clefting. Diehl et al. further teach the modulation of the clefting phenotype by a non-genetic factor that is a drug exposure and an interaction between two or more non-genetic factors that are drug exposures. The reference reports the findings of a genome-wide search for susceptibility genes for teratogen-induced clefting in the AXB and BXA set of recombinant inbred mouse strains, as they compare the results and the interaction between phenytoin(which induces cleft lip) and 6-aminonicotitinamide(which indices cleft palate) and the cleft palate phenotype(abstract and page 5231). The reference also teaches the method of a non-genetic factors ability to modulate the clefting phenotype wherein the phenotype is modulated by environmental, non-genetic factors such as a fctus' exposure in utero to ethanol, trimethadione, aminopterin and retinoic acid(page 5231). Included then in these findings are the reference's teachings of the identification of an interaction among two or more non-genetic factors(both environmental and drug-like) and a genetic locus. Furthermore, as stated previously, this same identification was made among multiple genetic loci discovered in this study in addition to those gene mutations that are well known in the art that the present reference reiterates, such as *Msx1*, several *Hox* genes, retinoic acid receptor alpha locus etc,(page 5231).

Response to Arguments:

Applicant's arguments filed 7/6/2004 have been fully considered but they are not persuasive. Applicant's first argue that Diehl "discloses standard recombinant inbred strains. Which one of ordinary skill in the art understands to be strains of mice that are homozygous at every locus(i.e. not genetically polymorphic)". However, Diehl's teaching of congenic strains does teach heterozygosity as congenic inbred strains of mice are as genetically like the mice of an existing inbred strain as possible in all respects, EXCEPT for alleles at one known locus at which two to four alleles have been deliberately kept segregating(ie. heterozygosity has been forced upon a particular locus. Applicant next argues that the disclosure of the A/WySn strain does not support a rejection under 102 because the strain is not a recombinant inbred(RI) strain. However, it should be noted that the limitation of being a RI strain is not required in claim 1 or claim 2, it is only one of the alternatives from claim 2. The applicant next argues that even if a RI strain is deemed to be present, "this disclosure does not teach backcrossing a recombinant inbred strain as recited in claim 1 to genetically map a locus associated with a phenotype"(Pg. 13 response). However, it is not clear where in claim 1 this limitation exists. Limitations in applicant's arguments, specification etc cannot be read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993). Furthermore, without a requirement for a RI strain in the claims, the art will be applied as broadly as the claims are written. The courts have stated that claims must be given their broadest reasonable interpretation consistent with the specification *In re Morris*, 127 F.3d 1048, 1054-55, 44 USPQ2d 1023, 1027-28 (Fed. Cir. 1997); *In re Prater*,415 F.2d 1393, 1404-05, 162 USPQ 541, 550-551 (CCPA 1969); and *In re Zletz*, 893 F.2d 319, 321-22, 13 USPQ2d 1320, 1322 (Fed. Cir. 1989) (see MPEP 2111).

2. Claims 1-4 are rejected under 35 U.S.C. 102(b) as being anticipated by Bellamy et al, (Human Genetics, 1991).

Bellamy et al. teach a method for identifying a genetic locus that modulates a phenotype, the method comprising:

(a) providing a renewable population of diploid humans that are genetically diverse individuals; and

(b) mapping the genomes of individuals within the renewable population of genetically diverse individuals that display the phenotype, whereby a genetic locus that modulates the phenotype is identified(Entire document especially pg. 345).

Bellamy et al. further teach the above method wherein the renewable human population of genetically diverse individuals comprises a panel of cell lines derived from genetically diverse individuals(Pg. 341).

Response to Arguments:

Applicant's arguments filed 7/6/2004 have been fully considered but they are not persuasivc. Applicant's first argue that the reference fails to show certain features of applicant's invention, it is noted that the features upon which applicant relies (i.e., "phenotypc" and "mapping genomes" are not explicitly defines in their claims or specification). Even if there was an exemplary definition, the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993). Furthermore, it is noted that the following two definitions were found in the UC biotech website and show the applicability of the Bellamy reference.

Phenotype: visible characteristic or traits of an organism, like a plant or an animal. Bellamy's teaching of varying band sharing in inbred populations is a visible characteristic resulting from the cutting of a specific restriction enzyme and the subsequent running through an agarose gel. Genetic Mapping: determination of the relative locations of genetic information on chromosomes. Bellamy's teaching of the placement and size range of band sharing for first degree relatives in the Gaza family for example, meets the limitation of mapping the genome. The fact that the genetic locus modulates the amount of band sharing is a result of inbreeding populations, but the loci responsible for this apparent modulation between strains are the VNTR loci and specifically "four multi-locus probes, 33.6 and 33.15, (CAC)5 and M13 were used to produce DNA fingerprints of all or part of the Gaza family"(Pg. 343). The claim does not require that for example, a mutation in the genetic locus in some way alters the expression of the encoded protein causing a aberrant phenotype. As a result the various loci probed in this reference serve to teach the way in which genetic loci modulate the phenotype of band sharing.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

3. Claims 11-14 and 16-18 are rejected under 35 U.S.C. 103(a) as being unpatentable over Diehl et al. in view of Dindzans et al.(J. of Immunology, 1986) and in further view of Hedrich, Hans J.("Genetic Monitoring, 1981).

Diehl et al. teach a method for identifying multiple genetic loci for example, *Col2a1*, *Col1a1* and *Col3a1*(page 5235) that modulate the phenotype of facial clefting in mice. Diehl et al have performed a genome-wide search for loci contributing to susceptibility to teratogen-induced facial clefting in the mouse. AXB and BXA recombinant inbred(RI) lines derived from crosses between A/J and C57BL6/J strains were supplied by M. Nesbitt and the mice were then bred by intercrossing recombinant inbred lines and maintained in a colony at the University of Michigan(page 5232) as a renewable population of genetically diverse individuals. The reference teaches this study for identifying a genetic locus in the diploid mouse system wherein the inbred lines of the renewable population of genetically diverse individuals comprise less than about 100 strains, in one instance a BXD set of 26 RI lines is used(page 5234). Experiments were also performed using the AXB and BXA RI strains to evaluate both spontaneous and teratogen-induced clefting resulting in both visual and physiological phenotypes. The reference uses the extensive data on teratogen-induced clefting in the AXB and BXA RI lines collected previously with a genome wide collection of marker typings for these RI lines to study the effects of genetic polymorphisms segregating in the renewable population(page 5232, left column). Diehl et al. teach the resulting molecular phenotype of their mouse mutants with clefting phenotypes to include for example, eight collagen genes including an altered expression of one, *Col3a1*, which is normally expressed in the embryonic palate. The reference also teaches the method for identifying multiple genetic loci further comprising identifying two or more genetic loci that modulate the phenotype of clefting as seen on the reference's page 5235 in their explanation that in addition to *Col3a1*, two other genetic factors, *Col1a1* and a cyclic nucleotide phosphodiesterase gene are located on the same chromosome and are thought to together, be

possibly relevant to the role of cAMP in the etiology of cleft palate abnormalities(page 5235). Additionally, the reference teaches the implication of the tenascin C gene, an extracellular matrix protein, and several cell-signaling molecules which have been previously implicated in clefting. Diehl et al. further teach the modulation of the clefting phenotype by a non-genetic factor that is a drug exposure and an interaction between two or more non-genetic factors that are drug exposures. The reference reports the findings of a genome-wide search for susceptibility genes for teratogen-induced clefting in the AXB and BXA set of recombinant inbred mouse strains, as they compare the results and the interaction between phenytoin(which induces cleft lip) and 6-aminonicotitinamide(which indices cleft palate) and the cleft palate phenotype(abstract and page 5231). The reference also teaches the method of a non-genetic factors ability to modulate the clefting phenotype wherein the phenotype is modulated by environmental, non-genetic factors such as a fetus' exposure in utero to ethanol, trimethadione, aminopterin and retinoic acid(page 5231). Included then in these findings are the reference's teachings of the identification of an interaction among two or more non-genetic factors(both environmental and drug-like) and a genetic locus. Furthermore, as stated previously, this same identification was made among multiple genetic loci discovered in this study in addition to those gene mutations that are well known in the art that the present reference reiterates, such as *Msx1*, several *Hox* genes, retinoic acid receptor alpha locus etc,(page 5231).

Diehl et al do not teach the derivation of the RI lines from at least 3, 4 or 8 non-recombinant parent lines or that genetically diverse individuals will be a natural by product from the use of multiple parent strains.

However, Dindzans et al. teach that multiple parents are necessary for the breeding of mice in an attempt to map genes and in the elucidation of mechanisms of genetic control. Dindzans et al. teach "the mode of inheritance of susceptibility/resistance to mouse hepatitis strain 3 (MHV)-3 being determined by typing the set of AXB/BXA recombinant inbred (RI) strain derived from **resistant** A/J and **susceptible** C57BL/6J progenitors for susceptibility to infection as determined by the severity of live pathology". "The strain distribution pattern for susceptibility showed a discontinuous variation: one strain was fully resistant(like A/J), four strains were fully susceptible (like C57BL/6J), and 16 strains showed an intermediate degree of susceptibility"(page 2355). Accordingly, it has been suggested that strain-dependent susceptibility to MHV-3 reflects genetically controlled immune defects rather than differences in the non-genetic, in this case viral factor. It is important to note the need for parental strain diversity that the reference teaches as " the AXB/BXA RI strains used in these experiments were derived from susceptible (C57BL/6J) and resistant (A/J) progenitors representing extremes in disease" for the sole purpose of creating RI strains exhibiting distinct patterns of MHV-3 induced liver pathology, and a discontinuous strain distribution pattern of S/R was seen(page 2357, discussion). This reference then teaches the importance of having an "unique assortment of parental genes that are homozygous at every locus, as such strains are useful for the mapping of genes and restriction sites and in the elucidation of mechanisms of genetic control"(page 2355). The reference teaches that multiple progenitors were used to establish their population for the expected benefit that using multiple progenitors creates an "unique assortment of parental genes" which is "useful for the mapping of genes and restriction sites and in the elucidation of mechanisms of genetic control".

Dindzans et al. do not teach the derivation of the RI lines from at least 3, 4 or 8 non-recombinant parent lines.

Hedrich teaches the organization of breeding colonies from a founding colony made up of 8-10 breeding pairs. Hedrich teaches in his Chapter on "genetic monitoring" of the mouse in biomedical research, that the organization of breeding colonies should include propagation steps consisting of three groups: "foundation colony (FC), pedigreed expansion colony (PEC), and production colonies(PC)"(Chapter 8, Page 171). Hedrich further teaches that the "foundation colony, which preserves the germline, should be of limited size" and that it may be either be built up as a single line (SL) or in a modified parallel line(MPL) system. With the SL system, Hedrich teaches that "SL colony members are usually more closely related to each other". In contrast, Hedrich teaches that "in the MPL system e.g. three family lines are kept for four generations, each consisting of not more than 8-10 breeding pairs"(Pg. 171). The reference continues to teach that "one breeding pair of the foundation colony is selected as common ancestor, whose offspring will again give rise to three family lines" and further that, "the degree of kinship is varying from generation to generation within the cycle". The reference teaches that this method makes "it possible to select among the lines that one which matches the original standards best".

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have modified the identification of a genetic locus that modulates a phenotype method of Diehl et al. so as to have included the diverse population of non-recombinant, parent lines of Dindzans et al. and to have derived their breeding population from at least 3, 4, or 8 non-recombinant parent lines as taught in further view of Hedrich, not only for

the expected benefit that more parents would obviously result in a more diverse progeny, but also for the expected benefit of providing an additional means for furthered variation among mouse lines and for the ability taught by Hedrich of making "it possible to select among the lines that one which matches the original standards best"(Page 171). Therefore, combining the teachings of Diehl et al. in view of Dindzans et al. and in further view of Hedrich would have been obvious at the time the invention was made.

Response to Arguments:

Applicant's arguments considering the deficiency of the cited art to meet the limitations of claims 1 and 2 are acknowledged and are addressed in the above sections of this action. It is is maintained that, with respect to applicant's arguments concerning this rejection that the "Dindzans reference does not disclose the production of RI lines from multiple(at least 3, 4, and 8) non-recombinant lines", and that Hedrich does not remedy this defect in Dindzans. Applicant is reminded that Dindzans was relied upon for it teachings of non-recombinant parental lines in its teaching of " the AXB/BXA RI strains used in these experiments were derived from susceptible (C57BL/6J) and resistant (A/J) progenitors representing extremes in disease" for the sole purpose of creating RI strains exhibiting distinct patterns of MHV-3 induced liver pathology. The reference thus teaches that multiple progenitors were used to establish their population for the expected benefit that using multiple progenitors creates an "unique assortment of parental genes" which is "useful for the mapping of genes and restriction sites and in the elucidation of mechanisms of genetic control". While Hedrich's teaching of 8-10 breeding pairs makes obvious the use of at least 3, 4, or 8 non-recombinant parent lines of Dindzans, not only for the expected benefit that more parents would obviously result in a more diverse progeny, but

also for the expected benefit of providing an additional means for furthered variation among mouse lines and for the ability taught by Hedrich of making "it possible to select among the lines that one which matches the original standards best"(Page 171). It should be noted that if applicant were to amend their claims to include the many limitations of their arguments, the art would have less applicability. As they are currently written however, there are not limitations read into them from the specification, arguments, etc. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993).

4. Claims 1 and 60-72 are rejected under 35 U.S.C. 103(a) as being unpatentable over Diehl et al. in view of Shire et al, (Genetical Research, 1989, 54. pp. 213-219).

While the teachings of Diehl et al. can be seen above, the reference does not teach the crossing or backcrossing of recombinant inbred lines.

However, with regard to claim 60, Shire et al. teaches paternal inheritance of egg traits in mice between reciprocal crosses of the CXBD and CXBE recombinant inbred strains but not in crosses between recombinant inbred strains with similar phenotypes(Abstract).

With regard to claims 61-63, as crosses involving C57BL/6ByEss and BALB/cByEss being crossed reciprocally to produce F1 and F2 hybrids and further that CXBD/ByEss, CXBE/ByEss and CXBJ/ ByEss reciprocal crosses of these RI strains were performed, the RI lines of claim 60 were derived from at least 8 different non-recombinant lines.

With regard to claims 64-72, Shire et al. teaches the phenotype of susceptibility of the cumulus oophorus and the zona pellucida surrounding the egg to enzymatic attack by hyaluronidase and protease(two or more non-genetic factors). "This paper describes the

phenotypes of eggs from females from the two reciprocal F1 crosses and four reciprocal F2 crosses between C57BL/6ByEss and BALB/cByEss, together with the results of a crossfostering experiment. Measurements on reciprocal F1 crosses involving some of the CXB recombinant in bred strains are also presented. The phenotype was modulated by a non-genetic factor that is a drug exposure in that ovulation was induced by injection of 8 i.u. pregnant mare gonadotrophin(PMSG: Sigma) followed by 8 i.u. of human chorionic gonadotrophin(hCG Boehringer-Mannheim)(Page, 214).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have modified the method for identifying a genetic locus that modulates a phenotype, by providing a renewable population of genetically diverse individuals produced by crossing or backcrossing recombinant inbred lines for the expected benefit of providing a more discriminating test between phenotypic models, as taught by Shier et al.; "the hyaluronidase sensitivity of the reciprocal crosses between inbred strains with similar phenotypes, whether susceptible or very resistant, did not discriminate between models". "The reciprocal F1 females from the cross of the CXBE(RI) and CXBD(RI) lines did differ significantly, and in the direction predicted by patroclinous inheritance" therein providing more discriminating potential between the two models and allowing for the comparison of the F1 females to be more similar to their BALB progenitor strain than to their immediate paternal strain.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sally A Sakelaris whose telephone number is 571-272-0748. The examiner can normally be reached on M-Fri, 9-6:30 1st Friday off.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Benzion can be reached on 571-272-0782. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Sally Sakelaris

9/2/2004

JEFFREY FREDMAN
PRIMARY EXAMINER
9/2/04